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Risperidone-induced sialorrhea responsive to biperiden treatment

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Background

Sialorrhea or hypersalivation is an uncomfortable adverse effect of antipsychotics. Salivary flow is predominantly under parasympathetic control, whereas the sympathetic system has a minor modifying effect in the saliva composition. However, the mechanism by which atypical antipsychotics cause hypersalivation remains controversial.

We report the case of a patient with risperidone-induced sialorrhea that responded well to I.M. administration of biperiden.

Materials and methods

The patient was an 18 year old female suffering from schizophrenia, paranoid type. She had no history of any medical condition. The onset of her psychiatric condition was at the age of 17. Due to low adherence, she had never taken any medications and had no previous hospitalizations. During her hospitalization, she was started on 1mg/d lorazepam and 2mg/d risperidone (with a gradual increase up to 6mg/d on day 3, due to lack of response and progressive thought disorganization). On day 4 the patient exhibited hypersalivation and a concomitant mild speech disturbance. No signs of EPS where present except for mildly impaired postural reflexes that could be considered as a sign of imminent EPS. Subsequently, risperidone was tapered to 3mg/d along with oral biperiden 2mg/d resulting to full remission of hypersalivation by day 5.

Results

Excessive salivation remains a paradoxical adverse effect of antipsychotic treatment possibly due to the antimuscarinic properties of several antipsychotics. The mechanisms proposed include a postsynaptic a-adrenergic-mediated, by blockade of the a-adrenoreceptors at the level of salivary glands, a cholinergic-specific M4 receptor stimulation and an abnormal deglutition by blockade of receptors in the pharynx or in the muscles involved in the swallowing reflex.

In our case, hypersalivation could be attributed to esophageal dysfunction, as an EPS, however there was no evidence of EPS. Moreover, sialorrhea occurred with risperidone that has no reported affinity for muscarinic receptors. The most likely mechanism seems to be through central a-adrenergic antagonism, since risperidone is a potent antagonist of a1 and a2-adrenoreceptors. On the other hand, biperiden probably reversed sialorrhea by re-establishing the adrenergic-cholinergic balance through muscarinic receptor blockade.

To our knowledge this is the first case report of a risperidone-induced sialorrhea responsive to biperiden administration. The data about the efficacy of anticholinergics in the treatment of antipsychotic-induced hypersalivation remain controversial suggesting the need for further investigation.